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TWO CASES OF ANEURYSM OF THE SPLENIC ARTERY.¹

(From the Pathology Department of Glasgow Royal Infirmary.)

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THE following cases are reported on account of their rarity as pathological conditions and the clinical features which they presented.

The first is a case of infective aneurysm occurring within the substance of the spleen in a boy of 14, who suffered from ulcerative endocarditis. The condition is extremely rare; indeed, we have been unable to trace any record of a similar case.

In the second, the pathological condition is less rare. Aneurysmal dilatation of the trunk of the splenic artery is, in a sense, only an exaggeration of the tortuosity and irregularity which is very common; but in the present case the occurrence of thrombosis had results of much interest both clinically and pathologically.

CASE I.—Male, æt. 14 years, was admitted to the Royal Infirmary on 9th March, 1910. Seven years previously he had an attack of acute rheumatism and was ill for about eight weeks. The parents knew nothing at that time of any cardiac involvement, and the recovery appeared to be complete. He had a second, but less severe, attack seven months

¹ Read at a meeting of the Glasgow Medico-Chirurgical Society held on 21st October, 1910. A large globular aneurysm of the splenic artery from the Museum of the Glasgow Royal Infirmary was also shown.

before admission—the feet, ankles, and knees being mainly affected. This attack lasted for about four weeks, and again he seemed to be quite well, but three months before admission he began to have irregular pains in various joints, and severe paroxysms of pain in the præcordial area. The paroxysms lasted for a few seconds only, but occurred very frequently and seemed to cause very acute suffering. This continued until admission, but he had no further severe pain during residence.

On admission to hospital, he was noted as being a puny, undersized boy; pale, and evidently having undergone rapid emaciation. He was able to lie down comfortably in bed. There was a purpuric eruption over the trunk. Arterial pulsation was visible in the vessels of the neck, and capillary pulsation was easily demonstrated.

The cardiac rhythm was regular, but the force of the systole varied somewhat. A systolic thrill could be felt over the apex of the heart.

The cardiac area was a little enlarged in all directions, but principally towards the left. Mitral systolic and double aortic murmurs were made out. Liver and spleen could be felt beneath the costal margin, but the spleen was excessively tender, and even the gentlest palpation could scarcely be borne. Urine contained much albumen, with granular and hyaline casts.

Throughout the night of 12th March he was very restless and complained of severe headache, and between 2 A.M. and 10 A.M. on 13th he had six short convulsive seizures, during each of which there was internal strabismus, tonic spasm of all the limbs, and loss of consciousness.

From 13th March he appeared to be making slow progress, until 2nd April, when he had another convulsive seizure in which he died.

Throughout residence his temperature ranged between 98° and 101°, the rise being in the evening, and only once did it reach 102·4°. Pulse ranged between 96 and 112 per minute. For thirty-six hours before death, the temperature ranged between 102° and 103°, and pulse 140 to 150.

Abstract from Post-mortem Journal: External.—The body was that of a greatly emaciated boy. There was considerable lividity of the lips and a slight general anasarca. No purpuric spots were visible.

Thorax.—The serous sacs were healthy. Lungs showed chronic venous congestion and were very œdematous.

Heart.—Both ventricles showed considerable hypertrophy and dilatation, with good healthy muscular wall. The aortic valves were incompetent. Large shaggy masses of vegetations in process of breaking down were present on all three aortic cusps. There was some old-standing thickening of the aortic and mitral valves, but no adhesion of the cusps. There was some thickening of the endocardium, extending down for nearly an inch from the aortic opening, and over this and the ventricular surface of the anterior mitral cusp were numerous recent vegetations. There was a ring of recent small vegetations on the lines of contact of the mitral valves. The pulmonic and tricuspid valves were healthy. There was a small aneurysmal dilatation at the commencement of the aorta, involving the orifice of the right coronary artery.

Abdomen.—The liver and kidneys showed the changes associated with chronic venous congestion. The spleen was greatly enlarged, with deep puckering of the surface and some perisplenitis. Half of the spleen was examined at once, and the remainder preserved for further investigation. The puckerings on the surface were cicatrices resulting from old infarctions. There was also a large recent hæmorrhagic infarction.

The portion of spleen examined contained several large masses of laminated thrombus, enclosed in cavities with definite smooth walls, and the cavities communicated with one another.

There were a number of enlarged caseous mesenteric glands, but neither ulcers nor tubercles could be found in the bowel. The mediastinal glands were also slightly enlarged, but not caseous.

Brain.—There was a little hæmorrhage into the pia-arachnoid over the right parietal region, and the whole right hemisphere showed very considerable bulging. In the bifurcations of almost all the smaller vessels at the base there were loose plugs, corresponding in macroscopical characters to the vegetations on the aortic valves. There was a small area of softening in the left frontal lobe. There was a very large hæmorrhage into the right cerebral hemisphere which had ploughed up the brain substance so much that its source could not be traced.

Films made from the spleen by direct smear showed large endothelial cells in various stages of disintegration, and a few polymorphonuclear leucocytes, but no organisms. Sections of the cardiac valves were also stained for organisms, with negative result.

The remainder of the spleen (see illustration) after preservation showed one large mass and several smaller masses of thrombus—the larger ones being laminated—apparently enclosed in dilated vessels. The large mass is clearly an aneurysm, and is related to the one remaining branch of the splenic artery, which remains at the hilus. This branch is about one-twelfth of an inch in diameter, and enters the spleen close to the large aneurysm, into which it passes directly. For about the last quarter of an inch of its course, just before it is lost in the large aneurysm, the vessel is slightly tortuous, and shows a very marked dilatation of its lumen. The large aneurysm extends along under the capsule for rather more than an inch, and ends in the cicatrix of an old infarction.

There is further a medium-sized mass of thrombus adjacent to the large mass which can be traced, gradually increasing in size, right through this half of the spleen. It occupies a large globular dilatation lying close beneath the capsule. Along its course it gives off several smaller branches, which were made out to be greatly dilated branches of the splenic artery filled with soft red thrombus. It appears to represent another, possibly the main, branch of the splenic artery.

Portions of the lining walls of the cavities, taken from different situations, were submitted to microscopical examination.

For the most part, the wall is composed of a definitely laminated structure, and this at first was taken to be the remnant of an altered and greatly distended arterial wall. At no part could any endothelial lining be found, and in sections stained by Weigert's method no elastic tissue could be demonstrated. Most probably, therefore, the wall was composed of the compressed and altered splenic tissue. Several sections showed a gradual transition from thrombus through the laminated structure of the wall, becoming more and more cellular until the ordinary appearance of the spleen was reached. The membranous wall consists of spindle-shaped cells with elongated nuclei lying with their long axis around the cavity. In some parts this is very dense, and the cells seem drawn out and closely packed together. The majority of the sections, however, show a more loose arrangement of the spindle-shaped cells, with varying degrees of round celled infiltration. The transition to splenic tissue is, in most instances, quite clear, the spindle cells losing their definite arrangement and outline, and giving the appearance of their having been derived

from the stroma of the spleen. In a few situations the thrombus abuts on splenic tissue, which shows much less alteration and could still be recognised as spleen, but these parts also show a very definite circular arrangement of stroma and cells suggesting pressure.

The etiology of the aneurysms can only be a matter of conjecture. The spleen itself showed the changes typical of chronic venous congestion. There was no evidence either in the spleen itself or in the walls of the cavities of any recent acute inflammatory condition, only a few polymorphonuclear leucocytes being seen.

The most feasible explanation, however, especially in a case of ulcerative endocarditis, is that their origin was inflammatory in nature. It is impossible to decide whether they were originated by infective emboli of the larger arterial branches or by organisms circulating in the blood and lodging in the spleen, there giving rise to a peri-arterial inflammatory condition such as has been described by Rolland in a case of aneurysm of the hepatic artery. The cicatrices of old infarcts go to prove the existence of fair sized emboli, and the reasonable presumption is that this has been the primary condition.

The small aneurysmal pouch at the commencement of the aorta did not show much change in the wall. The tunica media was a little thinner than elsewhere, and there was slight increase of fibrous tissue, but no sign of any active inflammation. This condition involving the orifice of the right coronary artery is interesting as an explanation of the severe paroxysms of cardiac pain.

No other vascular changes were found in any part of the body, but it is more than likely that the cerebral hæmorrhage resulted from rupture of an aneurysm, due in all probability to an infective embolism.

CASE II.—Female, æt. 43 years, was admitted to the Royal Infirmary on 3rd March, 1910. Her illness dated from twelve days previously, when she experienced a feeling of discomfort in the left hypochondriac region, but according to the history obtainable nothing definite was made out, and there was no fulness and no rigidity. The discomfort increased to actual pain, which she had almost continuously until admission. She was very constipated at first, but after taking purgative medicine she rather suffered from diarrhoea. The character of the stools does not seem to have been noted. She became much worse on the day before admission, and

vomited continuously, the vomitus being green coloured. Her pain was now severe, and there was slight distension of the abdomen, with rigidity of muscles above and to the left of the umbilicus.

On admission she was very collapsed, with small, rapid pulse, eyes sunken, and features pinched. She was noted as being rather cachectic, and of a lemon yellow colour. There was practically no rigidity nor distension of the abdomen, but there was a good deal of pain and tenderness in the epigastric and left hypochondriac regions. Hepatic dulness was not diminished. She was vomiting continuously, and the vomitus, besides being stercoraceous in character, contained altered blood.

She died two days later. There was no hepatic dulness on the day of her death. Her temperature ranged between 98.4° and 102° , the remission being in the evening. Two Widal's tests were performed before admission, on days reckoned to be seventh and fourteenth of illness, with negative result, but after admission to hospital a third examination in the sanitary office of the city of Glasgow was positive, and the case was notified as enteric fever prior to the *post-mortem* examination.

Abstract from Post-mortem Journal: External.—The body was that of a fairly well-nourished woman. There were no external markings.

Thorax.—Heart was small; valves were competent and healthy. Muscular wall was rather soft, and the fibres tended to separate. Aorta was practically healthy, and free from thrombi. Lungs were œdematous. The left was slightly adherent at the apex. The right was adherent all over, and very densely so at the base, where a new fibrous tissue formation enclosed a cavity, about the size of a golf ball, filled with pure white creamy substance.

Abdomen.—Almost the whole of the first half of the jejunum was intensely congested, and contained numerous small hæmorrhages. The first two coils (about 3 feet) were actually gangrenous. The bowel wall, except the gangrenous part, and mesentery were firm and considerably thickened. The affected coils of intestine were sealed together by recent fibrinous exudation, but otherwise there was no sign of peritonitis.

The stomach was apparently normal, and the duodenum was also normal looking for the first two-thirds of its length. Beyond that it was congested, but the real

infarction of the intestine began beyond the termination of the duodenum.

The main trunk of the portal vein was distended with rather dense thrombus, which passed far into its branches in the liver and down the mesenteric veins to the intestine. The cæcum and ileum showed no lesion of their walls and no sign of enteric ulceration, but their contents were blood-stained. There was thrombus lying loosely in the veins returning from the cæcum. The main trunk of the celiac axis and the hepatic artery were clear, but the splenic artery from immediately beyond its origin was thrombosed.

Spleen.—There was a large anæmic infarct occupying fully half of the organ. This was well defined at its margins, and very soft internally. It was of fairly recent origin, but definitely an older lesion than that of the intestines. The spleen was adherent to a much prolonged left lobe of the liver. The rest of the spleen showed rather full congested pulp.

Liver was pale, anæmic, and rather fatty.

Kidneys showed advanced cloudy swelling.

The only thrombi in the heart were agonal clots in the right side.

On further dissection, a remarkable condition of the splenic artery was made out. It followed a course to the spleen which was even more tortuous than normal, and it was distended with thrombus and showed globular dilatations which made it look like a thrombosed varicose vein. These were clearly shown by dissection to be aneurysmal dilatations of the artery.

The first of them—about half an inch in diameter—was situated about 3 inches from the origin of the vessel, and there were three others of irregular sacculated form, situated close to the hilus of the spleen.

The vessels entering the spleen were of the usual size. Most of them were occupied by red thrombus of varying density. The vessel at the apex of the large infarction contained a very firm grey plug. The aneurysms contained firm red thrombus, with little indication of a laminated structure.

Microscopical examination was made of sections, including several undilated arterial twigs at the hilus of the spleen and of portions of two of the aneurysms. The undilated twigs appear to have thickened walls, with a certain amount of endarteritis and fibrous changes in the middle coat. The walls of the aneurysms are fairly thick and very fibrous, with

a certain amount of round cells principally replacing the middle coat. The internal elastic lamina is not recognisable, and it would be scarcely possible to tell that they were of arterial origin apart from the dissection.

As to the course of events, only suggestions can be made. It seems probable that the aneurysms had long been in existence, and from the history it also seems probable that the occurrence of the large infarction corresponded with the onset of symptoms. This may have been due to embolism, in which case the embolus probably came from a thrombosis in one of the aneurysms; or it may have been due to direct occlusion of the arterial branch by thrombosis in an aneurysm.

The thrombosis gradually spread back to the commencement of the splenic artery, where it was arrested. About the same time thrombosis occurred in the splenic vein, which, however, extended into the main portal trunk, producing the thrombosis of the entire portal system, and giving rise to infarction of the intestine.

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REFERENCE.

W. Rolland, *Aneurism of the Hepatic Artery* (with review of literature), *Glasgow Medical Journal*, May, 1908.



